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TLR7 agonist imiquimod enhances the efficacy of 2009 pandemic H1N1 vaccine in BALB/c mice

Li Can
The University of Hong Kong, China

TLR ligands, which can directly stimulate immune responses towards antigens, have shown great promise as novel adjuvants in many vaccine studies. Imiquimod, a synthetic TLR7 agonist, could significantly enhance immune responses together with conventional vaccine strategies. Our previous study showed an inactivated vaccine combined with Imiquimod (VCI) could elicit immediate immune responses against influenza A (H1N1) pdm09 infection in BALB/c mice. Survival rate was significantly improved in VCI group. Neutralizing antibody and virus-specific antibody appeared earlier and with higher titer in VCI group. We further seek the mechanisms overlaying the enhancement of vaccine induced by Imquimod. Our recent study showed 18 hrs after intraperitoneal stimulation of VCI, B cells migrated rapidly from peritoneal cavity to spleen. CD86 on lymph node and splenic B cells was up-regulated in VCI group. After infection, B2 cells and T cells increased rapidly in lung of VCI group. Prompt neutrophil infiltration was also detected in lung, together with faster formation of germinal center around infection site. Higher affinity of local virus-specific antibody responses followed afterwards. These results indicated Imiquimod combined with vaccine may stimulate local B cells activation and enhance the proliferation and production of high-affinity antibody. Finally, the fast-acting antibody-based immune responses induced by vaccine combined with Imquimod may provide a new strategy facing immediate unpredicted influenza outbreaks. The mechanisms of early B cell activation deserve further study.

Biography

Li Can is a PhD candidate in the Dept. of Microbiology, the University of Hong Kong. She mainly carries out her researches on vaccine and the pathogenesis of influenza viruses, including pandemic H1N1 virus, H5N1 and H7N9 viruses. She has published several papers during her PhD study.

leecan10@gmail.com

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